Day : Monday
Date: 10/23/2006

PALM INTRANET

Time: 08:05:37

Biotech Query for 10/019067

Title: **DIAGNOSIS OF GLUTEN SENSITIVE ENTEROPATHY AND OTHER AUTOIMMUNOPATHIES**

Inventor: **PAULSSON, MATS**

Location:

Location Date: Group Art Unit: **1641**

Status: **71/RESPONSE TO NON-FINAL OFFICE ACTION ENTERED AND FORWARDED TO EXAMINER**

Barcode:

Num	Date	Code	Contents Description
34	10/04/2006	CRFF	ERROR(S) IN CRF CORRECTED BY STIC
27	08/14/2006	CRFD	CRF IS FLAWED TECHNICALLY / NOT ENTERED INTO DATABASE

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EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	1968	tgk or tgx or tge or (band adj "4. 2") or fxiiia	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 12:50
L2	56665	autoimmune	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 12:50
L3	74	1 and 2	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 12:55
L4	9	1 same 2	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 12:50
L5	251460	antibod$3 or iga or igg or igm or ige or autoantibod$3 or auto-antibod$3	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 12:56
L6	448	1 and 5	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 12:56
L7	89	1 same 5	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 13:12
L8	36	transglutaminase adj (k or x or e)	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 13:13
L9	31	5 and 8	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 13:22
L10	1336	gse or gse-type or (gluten adj sensitive adj enteropathy)	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 13:22
L11	3	1 and 10	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2006/10/23 13:22

FILE 'EMBASE, BIOSIS, CAPLUS, SCISEARCH, MEDLINE' ENTERED AT 12:39:05 ON 23 OCT 2006

L1	3065	S TGK OR TGX OR TGE OR (BAND (W) 4.2) OR FXIIIA
L2	3140	S GSE OR (GLUTEN (W) SENSITIVE (W) ENTEROPATHY)
L3	7393	S (DERMATITIS (W) HERPETIFORMIS) OR (MORBUS (W) DUHRING)
L4	1	S L1 AND L2
L5	3	S L1 AND L3
L6	1	DUPLICATE REM L4 (0 DUPLICATES REMOVED)
L7	21	S L1 AND AUTOIMMUNE
L8	16	DUPLICATE REM L7 (5 DUPLICATES REMOVED)
L9	716	S L1 AND (ANTIBOD? OR IGA OR IGE OR IGM OR IGG OR AUTOANTIBOD?)
L10	372	S L1 (S) (ANTIBOD? OR IGG OR IGA OR IGM OR IGE OR AUTOANTIBOD?)
L11	2	S L10 AND AUTOIMMUNE
L12	0	S L3 AND L10
L13	1	S GSE-TYPE

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	2660853	diagnos$ or determin$	US-PGPUB; USPAT	OR	ON	2006/10/24 07:50
L2	1262	(dermatitis adj herpetiformis) or (morbus adj duhring) or duhring	US-PGPUB; USPAT	OR	ON	2006/10/24 07:51
L3	1185	1 and 2	US-PGPUB; USPAT	OR	ON	2006/10/24 07:51
L4	89	1 same 2	US-PGPUB; USPAT	OR	ON	2006/10/24 07:51
L5	323	tissue adj transglutaminase	US-PGPUB; USPAT	OR	ON	2006/10/24 07:52
L6	13	4 and 5	US-PGPUB; USPAT	OR	ON	2006/10/24 07:52

FILE 'EMBASE, BIOSIS, CAPLUS, SCISEARCH, MEDLINE' ENTERED AT 08:21:10 ON 24 OCT 2006

L1	7692	S	(DERMATITIS (W) HERPETIFORMIS) OR (MORBUS (W) DUHRING) OR DUH
L2	13425097	S	DIAGNOS? OR DETERMIN?
L3	2784	S	L1 AND L2
L4	522	S	L1 (S) L2
L5	9214	S	TTG OR (TISSUE (W) TRANSGLUTAMINASE)
L6	33	S	L4 AND L5
L7	18		DUPLICATE REM L6 (15 DUPLICATES REMOVED)

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	4	recombinant adj human adj tissue adj transglutaminase	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:14
L2	18	human adj tissue adj transglutaminase	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:17
L3	653005	human	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:17
L4	382727	tissue	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:17
L5	2538	transglutaminase	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:17
L6	707	4 same 5	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:18
L7	266	3 same 6	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:32
L8	36	7 and (gluten or dermatitis or morbus or duhring)	US-PGPUB; USPAT; EPO	OR	ON	2006/10/25 09:33

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     FILE 'EMBASE, BIOSIS, CAPLUS, SCISEARCH, MEDLINE' ENTERED AT 14:35:28 ON
     24 OCT 2006
L5           206 S L1 AND ANTIBOD?
L6         16229 S L5 AND GSE OR COELIAC
L7           100 S L5 AND (GSE OR COELIAC)
L8            44 DUPLICATE REM L7 (56 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 14:39:11 ON 24 OCT 2006
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Note: Performing your original search, ***gse dermatitis herpetiformis antibodies***, in PubMed will retrieve *14* citations.

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All: 1 Review: 0

1: Clin Immunol. 2001 Mar;98(3):378-82. Related Articles, Links

ELSEVIER SCIENCE DIRECT

Tissue transglutaminase and endomysial antibodies-diagnostic markers of gluten-sensitive enteropathy in dermatitis herpetiformis.

Kumar V, Jarzabek-Chorzelska M, Sulej J, Rajadhyaksha M, Jablonska S.

IMMCO Diagnostics, Inc., Buffalo, New York, 14228, USA.

The association of Durhing's disease, commonly referred to as dermatitis herpetiformis (DH), with gluten-sensitive enteropathy (GSE) is supported by the presence of villous atrophy and endomysial antibodies (EMA). EMA are found to be a marker of GSE both in celiac disease (CD) and in DH. Since tissue transglutaminase (tTG) is believed to be the major autoantigen in GSE, the aim of our study was to determine the specificity and sensitivity of anti-tTG antibody ELISA compared to the EMA indirect immunofluorescence test. We studied 44 cases of DH, confirmed by the presence of IgA immune deposits in the dermal papillae, and 58 cases of CD conforming to the International Criteria of Diagnosing CD. The control group comprised 161 sera from patients with vesiculobullous disorders other than DH and 106 sera from normal healthy blood donors. Anti-tTG antibodies were detected in 36 of 44 DH (79%) and in 32 of 58 CD (55%) patients. EMA were positive in 33 of 44 DH (74%) and in 36 of 58 CD (62%) patients. Both the EMA and the antibodies to tTG were present in the majority of patients with DH and CD when they were on a normal gluten-containing diet and were absent when on a gluten-free diet for an extended period of time. There were, however, small discrepancies in positivity and negativity in tTG antibody-positive and EMA-negative patients and vice versa. There seems to be a correlation between the EMA titers and the anti-tTG antibody levels. This study confirms the high specificity and sensitivity of anti-tTG antibody ELISA for GSE and its strong correlation with EMA

both in CD and in DH. The results of anti-tTG antibody and EMA assays were comparable; however, in DH, tTG was somewhat more sensitive than the EMA test. For screening of DH, it is advisable to perform both EMA and anti-tTG antibody tests. Copyright 2001 Academic Press.

MeSH Terms:

- Autoantibodies/blood*
- Biological Markers
- Celiac Disease/diagnosis*
- Celiac Disease/immunology
- Dermatitis Herpetiformis/diagnosis*
- Dermatitis Herpetiformis/immunology
- Enzyme-Linked Immunosorbent Assay
- Humans
- Transglutaminases/immunology*

Substances:

- Autoantibodies
- Biological Markers
- Transglutaminases

PMID: 11237562 [PubMed - indexed for MEDLINE]

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ClinicalTrials.gov
PubMed Central

Note: Performing your original search, ***gse dermatitis herpetiformis antibodies***, in PubMed will retrieve *14* citations.

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All: 1 Review: 1

1: Dermatol Clin. 1990 Oct;8(4):759-69. Related Articles, Links

Dermatitis herpetiformis.

Otley C, Hall RP 3rd.

Department of Medicine, Duke University Medical Center, Durham, North Carolina.

The state of our understanding of the pathogenesis of DH relies on the integration of several key characteristics: (1) a high frequency of the HLA antigens HLA-B8, HLA-DR3, and HLA-DQw2, (2) an associated GSE, (3) the resolution of both the skin lesions and gut abnormalities in response to a gluten-free diet, and (4) the presence of granular deposits of IgA in normal and perilesional skin. The role of the HLA class II antigens expressed in patients with DH most likely relates to the afferent or initiating arm of the immune system. The association of the HLA-A1, -B8, -DR3, -DQw2 haplotype with Sjogren's syndrome, chronic hepatitis, Graves' disease, and other presumably immunologically mediated diseases, as well as the evidence that some normal HLA-B8, -DR3 individuals have an abnormal in vitro lymphocyte response to wheat protein and mitogens and have abnormal Fc-IgG receptor-mediated functions, suggests that this HLA haplotype or genes linked closely to it may confer a generalized state of immune susceptibility on its carrier, the exact phenotypic expression of which depends on other genetic or environmental determinants. It also is clear, from the association of DH with GSE and the ability to control the cutaneous manifestations of DH with a gluten-free diet, that the gut disease is a critical factor in the pathogenesis of DH. Several pathogenetic theories about the origin of the cutaneous IgA deposits in DH have been proposed, one of which states that the IgA is produced in the gut mucosa as a response to a dietary antigen or gut epithelial antigen and then cross-reacts with the skin of patients with DH. A second hypothesis is that the IgA produced in the gut binds to an antigen and is deposited in skin as an antigen-antibody complex. Finally, it could be that the gut mucosal abnormality simply

allows an unknown antigen access to the central immune system where an IgA antibody is produced that binds to skin. The failure to detect circulating IgA anti-basement membrane zone antibodies in patients with DH suggests that either the structures to which the IgA binds are not present in normal skin without DH, that IgA cannot bind to these structures in vitro, or that the circulating IgA is too scant for detection with conventional methods. Finally, it must be considered that the IgA deposited in DH skin may bind as a result of non-antigen-antibody interactions that cannot be duplicated in vitro.(ABSTRACT TRUNCATED AT 400 WORDS)

Publication Types:

- Review

MeSH Terms:

- Celiac Disease/immunology
- Complement C3/immunology
- Dermatitis Herpetiformis/immunology*
- Dermatitis Herpetiformis/pathology
- Dermatitis Herpetiformis/therapy
- Humans
- Immunoglobulin A/immunology
- Research Support, Non-U.S. Gov't
- Skin/immunology

Substances:

- Complement C3
- Immunoglobulin A

PMID: 2249367 [PubMed - indexed for MEDLINE]